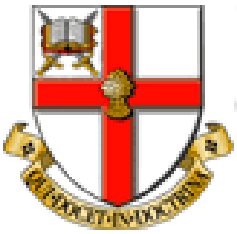

University of Chester

# Department of Clinical Sciences & Nutrition

## MSc :

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| Project Title | Lifestyle behaviours associated with type 2 diabetes risk in Australian construction workers. |

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## Declaration

This project contains no material that has been accepted for publication for the award of any other degree or diploma at any institution. I certify that all material in this assignment is my own work, except where I have indicated with appropriate references. The project does not contain experimental data from another person's work. The content of this project is the result of work that was carried out by me during my enrolment at the University of Chester. I agree that I will submit an electronic copy of this work for submission to a Plagiarism Detection Service for quality assurance purposes.

Signed SIAN BOTLEY

Date: 31/08/18

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I would like to thank the participants for completing questionnaires, and for the construction sites allowing us access to their workers. Also, I would like to sincerely thank my supervisor, Katherine Markwell, who has been extremely supportive and helpful throughout my research project.

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Tables and figures



Definitions and abbreviations

aMED – Alternative Mediterranean diet

AUSDRISK – Australian Type 2 Diabetes Risk Assessment Tool (Chen et al., 2010). A Questionnaire designed to estimate risk of type 2 diabetes.

BEVQ-15 – Questionnaire to estimate daily beverage intake (Hedrick et al., 2012).

BMI – Body mass index

CRF – Cardiorespiratory fitness

DASH – Dietary approaches to stop hypertension

DINE tool – Semi-quantitative food frequency questionnaire to determine fat intake (Roe et al., 1994)

FFA – Free fatty acids

FFQ – Food frequency questionnaire

HEI-2010 – Healthy eating index 2010

IPAQ – International physical activity questionnaire. Used to determine duration, frequency and type of physical activity (Craig et al., 2003)

PA – Physical activity

RCT – Randomised control trial

RR – Relative risk

SSB – Sugar sweetened beverage

T2DM – Type 2 diabetes mellitus

WHO – World Health Organisation

# Literature Review:

# Lifestyle behaviours and the association with type 2 diabetes risk

**Abstract**

Type 2 diabetes mellitus (T2DM) is a global problem with many unfavourable consequences. Obesity is the single largest predictor of T2DM. Additional modifiable risk factors include lifestyle behaviours such as poor diet and physical inactivity have also been identified to be key determinants of the disease, and are therefore key in delaying or preventing progression, as proven by many systematic reviews. The incidence of T2DM is increasing, despite efforts to reverse this trend, so barriers need to be identified and solutions proposed to aid individuals to achieve positive lifestyle behaviours. Habitual lifestyle behaviours can be determined by occupation and particular work stresses. The construction industry is a large working population in Australia whose health outcomes have not been fully explored in relation to T2DM risk. It is unknown if specific unfavourable lifestyle behaviours are adopted within this population which increase the risk of progression of this disease. This review will discuss the associated risk factors and how they can be modified to prevent progression of T2DM. A rationale will be proposed for further investigation of T2DM and its potential specific risk factors within the Australian construction industry.

## Introduction

Diabetes, globally, has a prevalence of 8.5% (World Health Organisation [WHO], 2017b). Of these cases 85-90% are T2DM (WHO, 2017b). In Australia, 1.7 million people are affected, with a further half a million estimated to be living with undiagnosed diabetes (Diabetes Australia, 2015). It is estimated that by 2040, 642 million people globally will develop T2DM (Cefalu et al., 2016).

Certain lifestyle behaviours have been linked with the causation and progression of T2DM (Wu, Ding, Tanaka, & Zhang, 2014). Little research has focused on occupation specific lifestyle behaviours which may be increasing an individual's risk of developing T2DM.

This knowledge is paramount in improving peoples' lives by identifying high-risk groups and providing occupational relevant information to reduce development of risk and the more serious consequences associated with the disease. This identification of high-risk individuals and implementing preventative strategies could also result in the reduced need for health service interventions, as well as increased economic productivity, in relation to reducing such risks (Schofield et al., 2017).

The specific areas to be considered are the effects of diet and physical activity (PA) in causation and prevention of T2DM, with a particular focus upon construction workers in Australia. They are a disadvantaged socio-economic group within Australia, whose specific health outcomes have not been investigated (Lingard & Turner, 2018). However, such factors within their working conditions may give rise to undesirable lifestyle behaviours, yet to be explored, potentially enhancing their risk of T2DM.

The present literature review will explore key modifiable risk factors associated with the risk of T2DM and how they can be modified in order to prevent progression of the disease. It also provides a rationale for further exploration of T2DM risk and lifestyle behaviours in Australian construction workers.

**Diagnosis and Consequences**

Diabetes is diagnosed by a resting blood glucose ≥ 7 mmol/l (WHO, 2017b) or a haemoglobin $A_{1c}$ ($HbA_{1c}$) reading >6.5% (WHO, 2011). Insulin resistance, a precursor for T2DM, is thought to have multifactorial causes including insulin signalling defects (Kolb & Martin, 2017), glucose transporter defects (Herman & Kahn, 2006), and lipotoxicity (Perry, Samuel, Petersen, & Shulman, 2014). Further progression of the disease causes alterations in insulin secretion due to β-cell dysfunction (Petersen et al., 2012; Kolb & Martin, 2017).

There are many consequences of T2DM. High blood glucose is the third largest predictor of premature mortality (Cefalu et al., 2016). Additional health complications associated with T2DM include increased cardiovascular risk, amputations, blindness and kidney failure (Wu et al., 2014; Samsom, Trivedi, Orekoya, & Vyas, 2016; Wang, Hess, Hiatt, & Goldfine, 2016). Microvascular complications such as retinopathy often occur with more extreme blood glucose elevation (Grundy, 2012; McLellan, Wyne, Villagomez, & Hsueh, 2014; Halban et al., 2014). These consequences can therefore have serious implications to an individuals' health.

There are great financial implications of T2DM. In the UK in 2010-2011, total associated costs are estimated at £23.7 billion in (Hex, Bartlett, Wright, Taylor, &

Varley, 2012) Whilst in Australia, the direct cost of T2DM was estimated to be $1507 million in 2008-2009 (Schofield et al., 2017). This did not incorporate indirect costs such as disability adjusted life years, lost productivity, lost earnings and home/work modifications (Schofield et al., 2017). The detrimental implications of diabetes therefore have a direct impact on the individual and the wider economy.

**Causes**

Whilst genetic factors can predispose an individual's likelihood of developing T2DM, this likely only accounts for a small proportion of an individual's risk (Prasad, & Groop, 2015). Most causes are modifiable risk factors (Wu et al., 2014; Uusitupa, 2018). A prospective study by Liu et al. (2016) identified lifestyle factors such as smoking, being overweight, physically inactive and having poor diet as important predictors of T2DM. Longitudinal and epidemiological studies found the single largest predictor of T2DM is being overweight or obese (Hu et al., 2001; Danaei et al., 2011; Kharroubi, & Darwish, 2015). Being obese or having a large proportion of visceral fat is linked to larger adipocytes, which themselves are insulin resistant, resulting in greater lipogenesis (Dedoussis, Kaliora, & Panagiotakos, 2007).

Those with T2DM exercise less, are more likely to have sedentary professions, had a greater contribution of fat to total energy intake and had a family history of diabetes (Imamura et al., 2013). Conversely, Hu et al. (2001) found individuals with a normal BMI, abstained from smoking, exercised regularly and consumed a diet high in fibre and low in saturated and trans-fat had the lowest relative risk (RR) of developing T2DM (RR = 0.09, 95% CI 0.05, 0.17). Hu et al. (2001) followed 84,941 female nurses for a

mean duration of 16 years. Approximately 90% of those which developed T2DM could be explained by sub-optimal lifestyle habits. A strength of the study is the sample size, allowing important relationships to be identified, and aid the development of lifestyle interventions. However, it is within females, and therefore unclear whether similar relationships exist in males.

These lifestyle factors will be explored in greater detail.

**Diet**

Fat

Several dietary factors have been associated with T2DM. Individuals with T2DM do not meet recommended guidelines for fruit and vegetable consumption (Burch, Ball, Somerville, & Williams, 2018). However, only 27% of adults (19-64 years) met the recommended guidelines of 5 portions of fruit or vegetables per day (Public Health England, 2016). In Australia, less than 5% of adults (19-64 years) achieved the recommended intake for vegetables and legumes (Australian Bureau of Statistics, 2016b). The recommended intake is five portions per day for females, and six for males (National Health and Medical Research Council, 2013). Therefore, this cannot infer causation, but may suggest meeting the recommendations may be protective.

High fat diets, particularly saturated fats, are a major risk factor for T2DM (Ortega, Berná, Rojas, Martía, & Soria, 2017). Those with T2DM, had a significantly higher proportion of energy from fat (30.2% ± 0.5%) when compared to healthy controls (27.8% ± 0.5%; $p < 0.001$) (Thanopoulou et al., 2003). There are also positive

associations between increased consumption of saturated fatty acids and insulin resistance, a precursor for T2DM (Gijsbers et al., 2016; Wallin et al., 2012).

The mechanisms by which fats induce insulin resistance are complex. Specific free fatty acids (FFA) may differentially alter insulin resistance (Hu et al., 2001). This is thought to be due to differences in the length of the chain, with longer chain FFA stimulating insulin secretion (Dedoussis et al., 2007).

In muscle, raised FFA inhibits insulin signalling leading to depressed insulin-stimulated muscle glucose transport potentially mediated by suppression of GLUT-4 translocation (Ortega et al., 2017). Additionally, elevated FFA in the blood stream provides more competition for energy substrate, resulting in less glucose taken up and utilised by the liver and muscles (Yu et al., 2002; Randle, Garland, Hales, & Newsholme, 1963). This leads to hyperglycaemia, also contributed to from increased endogenous glucose production in the liver (Tangvarasittichai, 2015). In summary, the main mechanisms have direct implications on the prevention of glucose uptake.

Sugar

A review by Stanhope (2016) concluded high sugar diets are positively linked to T2DM; largely due to the enhanced lipogenesis, further contributing to insulin resistance (Stanhope, 2016). Indirectly, consuming excess sugar can lead to weight gain, and ultimately obesity (Stanhope, 2016). It is postulated increased sugar in our diets over the past decades are largely due to increased added sugar, in the form of sugar sweetened beverages (SSB) (Malik & Hu, 2015).

High consumption of SSB e.g. fruit juices, energy drinks, have positive associations with T2DM (Wu et al., 2014; Imamura et al., 2015; Mozaffarian, 2016; Malik et al., 2010). Increased intake of SSB are also positively linked with greater adiposity and ultimately long-term weight gain (Hu, 2013; Mozaffarian, Hao, Rimm, Willett, & Hu, 2011). Weight gain has been estimated to be responsible for 50% of the mechanisms behind SSB increasing the risk of T2DM. Individuals who consumed 1 or more SSB per day had an 83% greater risk of T2DM, compared to those who consumed <1 SSB per month (Schulze et al., 2004).

Other

Further dietary components have been associated with T2DM including low fibre (Oretega et al., 2017), lack of vitamin D and calcium (Pittas, Lau, Hu, & Dawson-Hughes, 2007). However, these are beyond the scope of this review. Therefore, the composition of an individuals' diet can have significant impacts on T2DM risk.

Most studies use self-report methods to gain information on dietary habits e.g. 24-h diet recall and food frequency questionnaires (FFQ). Although these are relatively easy to complete and can be used in epidemiological studies to gain detailed information, they are liable to recall bias (Shim, Oh & Kim, 2014). They are also time-consuming, and potentially a burden to the individual, which may lead to underreporting (Skim et al., 2014). Very few studies account for measurement error associated with self-report dietary data and the magnitude of error appears to vary depending on the modality used to assess dietary habits (Kirkpatrick et al., 2014). Two studies concluded females and overweight individuals are more likely to underreport energy intake when

compared to doubly labelled water (Trijsburg et al., 2017; Lopes et al., 2016). More objective measures e.g. direct observation would be more accurate, but are more labour intensive, and can only be feasibly done in smaller samples (Kirkpatrick et al., 2014).

**Physical activity**

Physical inactivity can lead to weight gain, already highlighted as the most important risk factor for T2DM (Kharroubi, & Darwish, 2015). Low cardiorespiratory fitness (CRF) and physical inactivity are consistently identified as independent predictors of mortality (Lee et al., 2012; Sardinha, Magalhães, Santos, & Júdice, 2017). Globally, physical inactivity is thought to be responsible for 7% of the burden of T2DM (Lee et al., 2012). It can cause hyperinsulinemia, consequently increasing the risk of T2DM (Rockette-Wagner et al., 2015; Lahjibi et al., 2013).

The highest prevalence of inactivity has been observed in those who went on to develop T2DM, and it is estimated >533,00 deaths globally could be averted through reducing physical inactivity by as little as 10% (Lee et al., 2012).

A systematic review by Smith, Crippa, Woodcock, & Brage (2016) calculated a 26% risk reduction (95% CI 20%, 31%) in T2DM between those that reached PA recommendations (150 minutes moderate-intensity activity per week), compared to inactive individuals. Further risk reduction was observed with more PA: 300 minutes PA per week inferred a 36% risk reduction (95% CI 27%, 46%).

This review included 28 studies with over one million participants. A unique strength of this study was the use of marginal MET hours per week (MMET/h) of leisure time PA.

This better estimates energy expended through accounting for proportionality from resting metabolic rate, allowing greater comparison across individuals and studies (Smith et al., 2016). Another strength of this review was its heterogeneity, incorporating studies from a diverse range of countries. Nevertheless, within individual studies, most participants were from white populations, of high income. This may limit generalisability of the results. Additionally, inconsistencies in outcome measurements existed across studies for diagnosing T2DM, potentially leading to inaccurate results. A common limitation of PA studies is the self-report nature of acquiring data, which is liable to bias i.e. overreporting activities (Ainsworth, Cahalin, Buman, & Ross, 2015).

This systematic review is supported by other studies demonstrating a lower risk of T2DM in active individuals (Aune, Norat, Leitzmann, Tonstad, & Vatten, 2015; Wei et al., 2000; Siegal et al., 2009). It is therefore an important modifiable risk factor.

**Other risk factors**

Besides general weight gain, an additional modifiable risk factor is greater visceral fat (Kolb & Martin, 2017); more commonly known as an “apple” shaped body (Diabetes Australia, 2015). Higher visceral fat is more common in males (Nordström, Hadrévi, Olsson, Franks, & Nordström, 2016). It is hypothesized visceral fat may be a more important predictor of T2DM risk than body mass index (BMI) as it is not confounded by muscle mass (Kolb & Martin, 2017). Visceral fat is estimated indirectly through waist circumference (Kolb & Martin, 2017). Further studies on T2DM risk should incorporate visceral fat measurements when risk stratifying individuals.

Non-modifiable risk factors for T2DM include age, sex, family history and ethnicity: African, Asian, Australian Aboriginal and Torres Strait Islander Peoples and Africa-Caribbean at the highest risk (Hu, 2011; Australian Bureau of Statistics, 2016a). Males over the age of 65, with a family history of hyperglycaemia or diabetes are more likely to develop T2DM (Hu, 2011).

**Prevention**

Although treatments are available for T2DM, focus should be on primary prevention to reduce associated health complications: cardiovascular disease, amputations, kidney failure and blindness (Wu et al., 2014), in addition to lowering the economic burden on health systems and enhanced productivity (Schofield et al., 2017).

Preventative methods are particularly relevant for overweight or high-risk individuals. High-risk can be defined as a fasting blood glucose of 6.1-6.9mmol/L or 2h post oral glucose tolerance test (75g) of 7.8-11 mmol/L (Diabetes Australia, 2016). These individuals may be considered metabolically healthy: overweight or obese individuals, with no major metabolic disturbances (Hamer, Batty, & Kivimaki, 2012). A meta-analysis by Bell, Kivimaki, & Hamer (2014) concluded metabolically healthy obese individuals were at a significantly higher risk of developing T2DM compared to metabolically healthy normal-weight individuals (RR 4.03 95% CI 2.66, 6.09). This provides further support for the significance and importance of reducing body weight to prevent T2DM.

Many questionnaires and scales have been designed to estimate T2DM risk. Of relevance is the Australian Type 2 Diabetes Risk Assessment (AUSDRISK) tool (Chen et

al., 2010). This incorporates many risk factors previously identified, with specific questions relevant for Australian populations e.g. ethnic groups. 38 is the maximum score, and a score ≥12 indicates high-risk of developing T2DM in the next five years.

Lifestyle

Lifestyle interventions are at the forefront of effective preventative strategies with a focus on losing weight, improving diet and increasing PA (National Institute for Health and Care Excellence, 2017; American Diabetes Association, 2014). Numerous randomised controlled trials (RCT) have concluded positive effects of lifestyle interventions on reducing T2DM risk in individuals who are at high-risk (Albright & Gregg, 2013; Li et al., 2008; Lindström et al., 2013; Tuomilehto et al., 2001; Ramachandran et al., 2006; Gong et al., 2011). These have recruited large sample sizes (range 522-2766 participants), and its effects have been consistent across numerous countries. They also used long follow ups; for example, Li et al. (2008) found the lifestyle group had a 41% lower incidence of T2DM compared to the control group at 14 years post-intervention.

A recent systematic review by Kerrison et al. (2017) concluded lifestyle interventions effective in preventing or delaying the onset of T2DM in high-risk individuals. They also found intervention groups reverted to normoglycemia at a quicker rate and lost greater percentages of weight than the control groups. The greatest changes were observed in the short-term, with only modest changes observed long-term. However, there was still a difference from baseline, suggesting even small changes are beneficial in delaying disease progression.

This review (Kerrison et al., 2017) included nine RCT, deemed not to have a high-risk of bias, from a variety of countries. Sample size ranged from 88 to 2161 participants, and all participants had either impaired fasting glucose or impaired glucose tolerance. A strength of this study was its use of objective measures to quantify high-risk. All but one study had a lower cumulative incidence of T2DM at the end of the study. The authors postulated this could be explained by the group-based nature of the intervention not being as effective as individualised sessions and the short duration of follow up (six months). Large variety in methodology and delivery of interventions makes comparisons and conclusions regarding the most efficacious methods difficult. The diminished benefits observed long-term in all studies suggest motivation to change should be explored, to sustain and maintain initial positive changes.

These results are supported by other systematic reviews exploring lifestyle interventions to reduce T2DM risk (Yoon, Kwok, & Magkidis, 2013; Balk et al., 2015; Howells, Musaddaq, McKay, & Majeed, 2016; Gong et al., 2015). One major limitation of lifestyle interventions is initial financial and logistical costs associated with implementing such intensive supportive interventions on a nationwide scale (Li et al., 2015). However, the longer-term cost saving associated with reduced medications and hospitalisations due to complications of T2DM outweighs this initial cost (Li et al., 2015).

Diet

Whilst lifestyle interventions aim to utilise diet to create energy deficits for weight loss, they are also crucial for optimum dietary composition (Hu, 2011). The

Mediterranean diet, more famously known for its cardiovascular protective effects, has also demonstrated beneficial effects on T2DM risk (Wu et al., 2014; Georgoulis, Kontogianni, & Yiannakouris, 2014; Salas-Salvadó et al., 2015; Jacobs et al., 2015). The diet promotes high intakes of legumes, whole-grain cereal, dairy, nuts, fish and red wine (Wu et al., 2014). Compared to other modern diets such as low-fat, low-carbohydrate and vegetarian diets, the Mediterranean diet was concluded by Schwingshackl, Chaimani, Hoffman, Schwedhelm & Boeing (2018b) as the most efficacious for glycaemic control. This is hypothesised to be due to the high polyphenol content of the diet, namely fruit, vegetables and high fibre foods (Ortega et al., 2017). Schwingshackl, Missbach, König & Hoffman (2015) performed a meta-analysis and found adherence to a Mediterranean diet resulted in a 19% risk reduction of developing T2DM. This suggests even those who have no current risk factors can still improve future health status.

This meta-analysis incorporated nine studies (n = >100,000). Adherence was measured in accordance with Trichopoulou, Costacou, Bamia, & Trichopoulos (2003) using a nine-point scale representing the nine key components of the Mediterranean diet. Semi-quantitative FFQ were used to assess dietary intake. However, food intake questionnaires are highly vulnerable to response bias (Probst, & Zammit, 2016). Additionally, one study recruited participants with a history of gestational diabetes, while another recruited participants with prior myocardial infarction, potentially increasing their risk of T2DM compared to a healthy population at baseline. This could have overestimated the beneficial impact of the diet.

Other dietary patterns are effective in reducing T2DM risk in healthy populations. Cespedes et al. (2016) found a 10-14% risk reduction in T2DM in individuals without hyperglycaemia who achieved a 1-SD increase in any of the four dietary indices assessed: Alternate Mediterranean Diet [aMED], Heathy Eating Index 2010 [HEI-2010], Alternate Healthy Eating Index, Dietary Approaches to Stop Hypertension [DASH].These diets all have commonalities, namely fruits, vegetables and whole-grains, but these results are important as there are multiple ways to achieve high scores on each index, suggesting diets can be tailored to individual preferences. This was a large prospective cohort study incorporating more than 100,000 ethnically diverse female participants from the Women's Health Initiative, followed up for a median of 15 years. Incidence of T2DM was self-reported, which may limit the accuracy of the results. However, a recent systematic review also reported similar benefits in T2DM risk in healthy individuals (Schwingshackl, Bogensberger, & Hoffmann, 2018). This review incorporated 1,670,179 participants of both sexes, providing strong evidence for the beneficial impact of optimal dietary habits.

Considering dietary composition further, most diets recommend limiting saturated fat intake and increasing polyunsaturated and monounsaturated fat intake (Wu et al., 2014). This is supported by studies finding no relationship between total fat and T2DM risk; but highlighting that the composition of dietary fat may be more important (Ley, Hamdy, Mohan & Hu, 2014). Despite no convincing evidence of a link between saturated fats and T2DM, replacing saturated fats can have substantial health benefits (De Souza et al., 2015).

A systematic review by Schwab et al. (2015) concluded probable evidence for replacing saturated fats with monounsaturated fats for improvements in fasting insulin concentrations and insulin sensitivity. Moreover, a systematic review and meta-analysis by Imamura et al. (2016) concluded replacing saturated fats with polyunsaturated fats lowered glucose and $HbA_{1c}$. Although monounsaturated fats did not alter fasting glucose, they did reduce $HbA_{1c}$ when compared to saturated fats (Imamura et al., 2016). An increase in energy of 5% from either monounsaturated fats or polyunsaturated fats corresponded to a 0.1% reduction in $HbA_{1c}$ (Chamnan et al., 2011). Although small, this clinically significant change translated into a 22% risk reduction (Chamnan et al., 2011). This is supported by a pooled analysis conducted by Wu et al. (2017) who concluded a high intake of polyunsaturated fats, particularly omega-6 fatty acids, resulted in a 43% relative risk reduction in T2DM in those who did not have diabetes at baseline.

A key target for primary prevention of T2DM is SSB due to its high consumption rate in younger individuals, which can translate into continued high consumption and poor health choices in later life (Singh et al., 2015). As previously mentioned, SSB have a strong association with obesity (Hu, 2013), and are consequentially associated to T2DM (Imamura, 2015). Reducing intake of SSB or swapping for alternative drinks can have positive health benefits (Pan et al., 2012). The Nurses' Health Study found that, while water consumption was not associated with risk of T2DM, replacement of one SSB with water was significantly associated with a 7% (95% CI 3%, 11%, $p < 0.05$) lower risk of T2DM (Pan et al., 2012). However, there could be some misclassification of diabetes as it was self-reported diagnosis.

Similarly, the replacement of SSB with water has also been documented to have positive effects on BMI in metabolically healthy individuals (Ebbeling et al., 2012; De Ruyter, Olthof, Seidell, & Katan, 2012). However, both studies recruited children and adolescents, so may not be reflective of adults. Artificial sweeteners have also been postulated as an alternative for SSB (Mozaffarian, 2016). Although, they are recommended only as an intermediate to help reduce intake of SSB, as they have been associated with potential health issues such as cognitive processes, dysfunctional energy homeostasis and altered taste receptors (Pepino, 2015).

Limitations of most studies is they are short duration, not randomised, and often do not assess endpoints e.g. incidence of T2DM, but rather measure insulin and glucose biomarkers (Imamura et al., 2016). Also, diet studies often use self-reported questionnaires to assess intake which are inherently limited by their subjective nature, and liable to underreporting and response bias (Probst, & Zammit, 2016).

Linked to this, the Hawthorne effect may be present in studies, as individuals may alter their behaviour due to the nature of being observed (McCambridge, Witton, & Elbourne, 2014). This could result in inaccurate and sometimes conflicting results, especially those with short duration, where significant changes have been achieved, although the magnitude of this effect is unknown (McCambridge et al., 2014). This can lead to misleading conclusions to be drawn regarding the efficacy of certain diets and their effect on T2DM risk (Kolb & Martin, 2017). Laboratory studies would be required to accurately assess biophysiological changes in insulin sensitivity in rigorously controlled settings. Further research is needed with longer follow-ups to determine

the longevity of biochemical effects, and assess compliance to diets, alongside strategies to enhance adherence.

Physical activity

Another important factor in T2DM prevention is PA. The Australian Government Department of Health (2017) recommends 150 minutes of moderate-intensity activity per week, in line with other governments and professional organisations (American Diabetes Association, 2017; UK Chief Medical Officers' Guidelines, 2011).

A systematic review by Smith, Crippa, Woodcock, & Brage (2016) concluded a negative correlation between PA and incidence of T2DM. Additional benefits can be accrued through exceeding the recommendations (Smith et al., 2016). This is supported by further studies demonstrating a protective effect of PA for developing or delaying T2DM (InterAct Consortium, 2012; Colberg et al., 2010; Fretts et al., 2009; McCarthy et al., 2017; Lavie et al., 2004). This effect is thought to be independent of genetic and familial influences, as highlighted in a twin study by Waller et al. (2010).

A study by McCarthy et al. (2017) recruited 489 individuals defined as being high-risk from their score on Leicester Practice Risk Score (LPRS). The LPRS is a questionnaire designed to identify adults at high-risk of developing T2DM aged 40-75 (Gray et al., 2012). They found increasing PA by 30 minutes per day resulted in significantly lower $HbA_{1c}$ (β = -0.14% 95%CI -0.20, -0.08); and increases in body weight resulted in significantly higher $HbA_{1c}$ (β = 0.08% 95% CI 0.04, 0.12). Despite these changes appearing unremarkable, these individuals did not have T2DM at baseline, therefore, smaller changes would be expected compared to those with the disease. This is

supported by stronger associations with changes in $HbA_{1c}$ found in those who exhibited hyperglycaemia at baseline. A strength of this study was that it objectively measured PA using accelerometers; eliminating bias associated with self-reported questionnaires. However, this was an observational study, and further research should incorporate RCTs to elucidate these results.

Regular PA achieves acute and chronic benefits which act to prevent hyperglycaemia. Exercise can acutely activate glucose transport and improve insulin sensitivity up to 48 hours post-exercise (Sylow, Kleinert, Richter, & Jensen, 2017). Chronic effects of exercise include increased muscle mass, improved insulin action, glucose control, fat oxidation and storage (Colberg et al., 2010).

There is evidence suggesting PA and CRF may be independent risk factors for T2DM (Lavie et al., 2014). Lavie et al. (2014) concluded an inverse relationship existed between mortality in patients with T2DM and both CRF and PA. Primarily, there appeared to be a substantial increase in mortality in those with low CRF. PA in this paper was regarded as reducing sedentary time. Recent studies suggest that without changes to purposeful exercise, purely breaking up sedentary time can have positive effects on insulin sensitivity and glycaemic responses (et al., 2017; Dempsey, Owen, Yates, Kingwell, & Dunstan, 2016; Chastin, Egerton, Leask, & Stamatakis, 2015). However, the effect of this only appears to be present in individuals with dysregulated glycaemic control, and not metabolically healthy individuals (Dempsey et al., 2016). Despite this, it still may be a useful recommendation for the primary prevention of T2DM.

**Construction industry**

Construction accounts for 6.5% of the workforce in the UK (Office of National Statistics, 2018), and is the third largest employment industry in Australia, employing over 10 million people (Parliament of Australia, 2016). Construction workers are considered a disadvantaged socio-economic group due to unfavourable working conditions, long working hours, levels of education, wealth and assets (Lingard & Turner, 2018; Naveena, 2016). Consistent relationships are observed between low socio-economic status and health outcomes (Kolmet, Marino, & Plummer, 2006).

Most health and safety research within the construction industry has focussed on the latter, and how to prevent injuries among its workers (Sherratt, 2018; Järvholm et al., 2014). Whilst this is a prominent area of interest and plays an important role in an individuals' competency to perform their job, less research has focussed on the main causes of early retirement witnessed in this population: disability and poor health (Hengel et al., 2012; Eaves, Gyi, & Gibb, 2016).

Relationships between working conditions and certain cancers have already been documented e.g. asbestos with lung cancer, sun exposure with skin cancer (WHO, 2017a). Other major health conditions in this populations, including T2DM, have been largely neglected in the research. This is especially important when evaluating the indirect costs of T2DM. It is estimated in 2012 in Australia, that lost labour force participation accounted for $384 million (Schofield et al., 2017). This translated to 4.2% of 45-64 year olds being out of the labour force due to diabetes in 2010; projected to increase to 4.95% in 2030 (Schofield et al., 2017). Additionally, most construction

workers are male, which as previously explored, are at greater risk of developing T2DM and are more likely to have greater visceral fat (Nordström et al., 2016; Hu, 2011). This highlights the importance of exploring whether this population could be considered at high-risk of diseases, and worthwhile of further investigation into enhancing their health outcomes.

Dietary and PA behaviours adopted by construction workers are currently unknown, and how this may impact upon T2DM risk has not been estimated. Due to the physically demanding nature of the job, it is unknown if individuals engage in purposeful exercise outside the work environment. A recent study suggested that caffeine consumption, is common within this environment, and could potentially be a health hazard (Loudoun & Markwell, 2017). This study was the first investigation of lifestyle behaviours adopted in the construction industry. However, further research is required in this population to interrogate other lifestyle behaviours which could have potentially detrimental health consequences.

**Conclusion**

A multitude of research has proved that, to some extent, dietary and PA factors can have an impact on an individual's risk of developing T2DM. Various studies have been undertaken but specific workgroups and occupations have not been investigated. Construction workers in Australia forms a large proportion of the employment industry and the potential economic, financial and health implications associated with a high incidence of T2DM in this group could be vast. Therefore, it is important to identify

whether this population is at risk, and if present, strategies should be implemented to prevent further progression based on the lifestyle factors identified.

The aim of this study is to firstly calculate the T2DM risk of Australians employed within the construction industry. Subsequently, the relationship between this risk will be explored with these key lifestyle habits: SSB intake, fat intake and PA to determine which risk factors are the most associated with increased likelihood of disease progression within this population.

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# Research study:

# Lifestyle behaviours associated with type 2 diabetes risk in Australian construction workers.

**Key words: physical activity, diet, occupation, health**

The Australian and New Zealand Journal of Public Health is the journal this study aims to be published in. Type 2 diabetes is a major health problem globally. Occupation specific lifestyle behaviours and health outcomes have often focussed on sedentary jobs or shift-based work. Other occupations have largely been neglected. In particular the construction industry, which constitutes the third largest employer in Australia (Parliament of Australia, 2016). Addressing potential health and lifestyle adversities in this population has important widespread public health implications, for improving individuals' quality of life, enhancing work productivity and relieving burdens on the health systems.

Abstract

*Background:* Despite type 2 diabetes mellitus (T2DM) being largely preventable through lifestyle modifications, the incidence is rising. Construction workers lifestyle behaviours are largely unknown, despite being Australia's third largest employer. *Aim:* Estimate construction workers' T2DM risk and explore relationships with lifestyle variables: total fat, unsaturated fat, sugar sweetened beverages (SSB) intake and physical activity (PA). *Methods:* 250 construction workers answered demographic and lifestyle behaviour questions (BEVQ-15, DINE, short-IPAQ, AUSDRISK tool). 228 participants were included for analysis: ANOVA, correlation and linear regression (35.3 years ± 11.7 years, BMI 27.1 ± 4.7). *Results:* 16% of participants were considered high-risk of developing T2DM. Low-risk individuals performed significantly more PA per week compared to medium-risk ($p = 0.012$), and high-risk individuals ($p < 0.01$). A significant linear correlation and linear regression also existed between PA and AUSDRISK score ($r(226) = -0.32$, $p < 0.01$,$F(1,226) = 6.464$, $p = 0.012$ respectively). A small negative relationship was detected between unsaturated fat and AUSDRISK score ($r(206) = -0.176$, $p = 0.011$). No significant results existed with total fat or SSB. *Conclusion:* High-risk individuals were older, had greater BMI, performed less PA and consumed less unsaturated fat. Strategies should target these key variables, to prevent progression of T2DM, to benefit the individual and economy.

## Introduction

Diabetes, globally, has a prevalence of 8.5% (World Health Organisation [WHO], 2017). In Australia, 1.7 million people are affected, with a further half million estimated to be living with undiagnosed diabetes (Diabetes Australia, 2015). Of these cases 85-90% are T2DM (WHO, 2017b).

There are many consequences of T2DM. Health complications include increased cardiovascular risk, amputations, blindness and kidney failure (Wu, Ding, Tanaka, & Zhang, 2014; Samsom, Trivedi, Orekoya, & Vyas, 2016). From an Australian economic perspective, direct costs were estimated to be $1507 million in 2008-2009 (Schofield et al., 2017). Whilst genetic factors can predispose individuals to T2DM, most causes are modifiable (Wu et al., 2014). Longitudinal and epidemiological studies have found being overweight or obese is the largest predictor of T2DM (Hu et al., 2001; Danaei et al., 2011; Kharroubi, & Darwish, 2015). Higher visceral fat is also a risk factor for T2DM (Kolb & Martin, 2017), which is more common in males (Nordström, Hadrévi, Olsson, Franks, & Nordström, 2016). Other important modifiable lifestyle factors have been identified in a prospective study by Liu et al. (2016) include poor diet, physical inactivity and smoking.

Lifestyle alterations can reduce the incidence of T2DM as concluded by many systematic reviews (Kerrison et al., 2017; Yoon, Kwok, & Magkidis, 2013; Balk et al., 2015; Howells, Musaddaq, McKay, & Majeed, 2016). The Australian Type 2 Diabetes Risk Assessment (AUSDRISK) tool (Chen et al., 2010) estimates T2DM risk, incorporating key risk factors including lifestyle and non-modifiable risk factors, with

specific questions relevant for Australian populations e.g. Indigenous heritage. It can be useful to gauge individual and population risk of developing diabetes.

Construction is the third largest employment industry in Australia, employing over 10 million people (Parliament of Australia, 2016). Construction workers are considered a disadvantaged socio-economic group due to unfavourable working conditions, long working hours, levels of education, wealth and assets (Lingard & Turner, 2018). Consistent relationships exist between low socio-economic status and health outcomes (Kolmet, Marino, & Plummer, 2006). Little research has focussed on causes of early retirement in this population: disability and poor health (Hengel et al., 2012; Eaves, Gyi, & Gibb, 2016).

Type 2 diabetes is a major health condition unexplored in this population but is especially important when evaluating the indirect costs. It is estimated in 2012 in Australia, that lost labour force participation accounted for $384 million (Schofield et al., 2017). The impacts of T2DM can negatively affect an individual's capacity to work if undiagnosed, and therefore is an important area to research to enhance productivity and individuals' health (Samson et al., 2016).

Habitual dietary behaviours such as fat intake, sugar sweetened beverages (SSB) intake and physical activity (PA) are key determinants in the progression, prevention and treatment of T2DM; therefore, providing a significant area for exploration (Ortega et al., 2017). The dietary and PA behaviours adopted by construction industry employees are currently unknown, and how this impacts upon T2DM risk has not been estimated. A recent study suggested caffeine consumption is common within this environment

and could potentially be a health hazard (Loudoun & Markwell, 2017). Further potentially detrimental lifestyle behaviours have not yet been explored.

## Methods

### *Study Aims*

The aim of this study is to firstly calculate the T2DM risk of Australians employed within the construction industry. Subsequently, the relationship between this risk will be explored with key lifestyle habits: sugar sweetened beverages intake, fat intake and physical activity to determine which risk factors are the most associated with increased T2DM risk within this population.

### *Hypotheses*

The first experimental hypothesis is that higher SSB and total fat intake will be associated with a higher risk of T2DM. The second experimental hypothesis that that higher unsaturated fat intake and higher physical activity will be associated with a lower risk of T2DM.

### *Participants*

250 construction workers completed surveys. The data was collected by Griffith University employees in at six urban construction sites in Brisbane, Australia. Ethics was granted from Griffith University (this ethics has been approved at the University of Chester as appropriate) and informed consent was provided by participants who completed the questionnaire.

### *Research Design and Measures*

A cross sectional study design was used and a questionnaire consisting of questions pertaining to demographic, occupational and lifestyle information was distributed during work breaks (Appendix 1).

Quantitative data on lifestyle behaviours (diet and physical activity) adopted by construction workers was also collected. Dietary information collected included the validated BEVQ-15 questionnaire (Hedrick et al., 2012), which had minor adaptations by research dietitian (Katherine Markwell) to reflect the Australian food supply and serve sizes, was used to estimate average daily SSB in kilocalories. The validated DINE tool (Roe, Strong, Whiteside, Neil, & Mant, 1994), a brief food frequency questionnaire, was used to gather information regarding unsaturated and total fat consumption. Total fat was classified into low (< 30), medium (30-40) or high (> 40) intake. Unsaturated fat was also classified into low (< 6), medium (6-9) or high (> 9) intake. Fruit and vegetable intake was collected using questions from the Australian National Health Survey. Ethnicity, age, sex and other questions were also asked. Questions regarding type, frequency and duration of physical activities undertaken were collected in the short International Physical Activity Questionnaire (Craig et al., 2003) and were compared with whether individuals met government recommendations of a minimum of 150 minutes cardiovascular activity per week (Australian Government Department of Health, 2017). Scales and tape measures were provided for participants to self-measure and self-report anthropometric variables (height, weight, waist circumference).

The AUSDRISK tool was used to evaluate an individual's risk of developing T2DM based on participants' responses to other survey questions, e.g. inputting combined fruit and vegetable intake, physical activity level, BMI. This questionnaire estimates the probability of developing T2DM in the next 5 years based on several risk factors: age, sex, country of birth, family history, history of high blood glucose concentration, hypertension, smoking status, fruit and vegetable intake, physical activity status and waist circumference (Chen et al., 2010).

Participants were excluded from analysis if two or more questions were unanswered.

*Statistical analysis*

Data was analysed using IBM SPSS Statistics (version 24). Descriptive statistics were used to report low, medium and high risk of type 2 diabetes in participants. Independent measures ANOVA's were conducted to compare the effect of total fat intake, unsaturated fat intake, SSB intake and physical activity on risk group. Post hoc analysis will be performed, if required, to explore where any differences may exist. These will be independent samples t-tests, or Mann Whitney tests if data is non-parametric. Significance values were adjusted by Bonferroni corrections to reduce type one error associated with multiple comparisons.

Correlations between relationship between AUSDRISK and the aforementioned variables. These will be Pearson if normally distributed, and Spearman rank correlations if data is non-parametric. Effect size will be determined using Cohen's (1992) guidelines. Linear regression analyses were used to see if the exploratory variable can predict AUSDRISK score. Only one variable was significant from the linear

regression, therefore multiple linear regression analysis could not be used to model the relationship between multiple variables.

Results

Data is reported as mean ± SD, unless otherwise specified. Significance was defined as $p < 0.05$, except for post hoc analysis of ANOVA where significance is set at $p < 0.17$.

*Participant characteristics*

228 participants were initially included in the analysis after the exclusion of incomplete questionnaires (35.3 years ± 11.7 years, BMI 27.1 ± 4.7, 0.024% females) (Appendix 2).

*AUSDRISK score*

Scores were found to be not normally distributed according to the Shapiro-Wilk value ($p$ <0.05) (Coakes & Steed, 2007); therefore, non-parametric analysis were performed (Appendix 3).

Table 1 shows 29.4% of participants were deemed low risk for T2DM (score ≤ 5). 54.4% were at a medium risk (score 5-11), and 16.2% at high risk (score ≥ 12).

A Kruskall-Wallis ANOVA was conducted to compare age and BMI between the T2DM risk groups. There was a significant effect of age at the $p < 0.05$ level for the three risk groups [$F(2, 191) = 61.03$, $p < 0.01$]. Post hoc comparisons using the Mann-Whitney test indicated that the mean age for the low-risk group (26.3 ± 6.7) was significantly lower than the medium-risk group (38.3 ± 11.5, $p < 0.01$) and the high-risk group (44.1 ± 9.4 $p < 0.01$). However, there was no significant difference in age between the medium and high-risk group ($p = 0.06$) (Appendix 4.1).

There was a significant effect of BMI at the $p < 0.05$ level for the three risk groups [F(2, 204) = 11.92, $p < 0.01$]. Post hoc comparisons using the Mann-Whitney test indicated that the mean BMI for the high-risk group (28.5 ± 3.6) was significantly higher than the medium-risk group (26.8 ± 4.3, $p = 0.006$) and the high-risk group (26.8 ± 5.3, $p = 0.003$). However, there was no significant difference in BMI between the low and medium-risk group ($p > 0.5$) (Appendix 4.2).

Table 1. Characteristics of participants in each risk group of the AUSDRISK tool[a]

| | Low risk (≤ 5)[a] | Medium risk (6 – 11) | High risk (≥ 12) |
|---|---|---|---|
| Participants (number, %) | 67 (29.4) | 124 (54.4) | 37 (16.2) |
| Age (years)[b] | 26.3 ± 6.7 | 38.3 ± 11.5* | 44.1 ± 9.4* |
| BMI[b] | 26.8 ± 5.3 | 26.8 ± 4.3 | 28.5 ± 3.6*[#] |

[a)] AUSDRISK tool (Chen, et al., 2010) estimates risk of diabetes in 5 years, total score in brackets

[b)] Data reported as mean ± standard deviation

*denotes sig difference from low-risk group ($p$ <0.01)

[#]denotes sig difference from medium-risk group ($p$ <0.01)

*Physical activity*

A Kruskall-Wallis ANOVA was conducted to compare PA in minutes per week between the T2DM risk groups (Appendix 5.1). There was a significant effect of PA at the $p < 0.01$ level for the three risk groups [F(2, 226) = 16.62, $p < 0.01$]. Post hoc comparisons using the Mann-Whitney test indicated that PA was significantly higher in the low-risk

group (825 ± 2180 minutes) compared to the medium-risk group (439 ± 897, $p = 0.012$) and the high-risk group (179 ± 331 minutes, $p < 0.01$). However, there was no significant difference in age between the medium and high-risk group ($p = 0.05$).

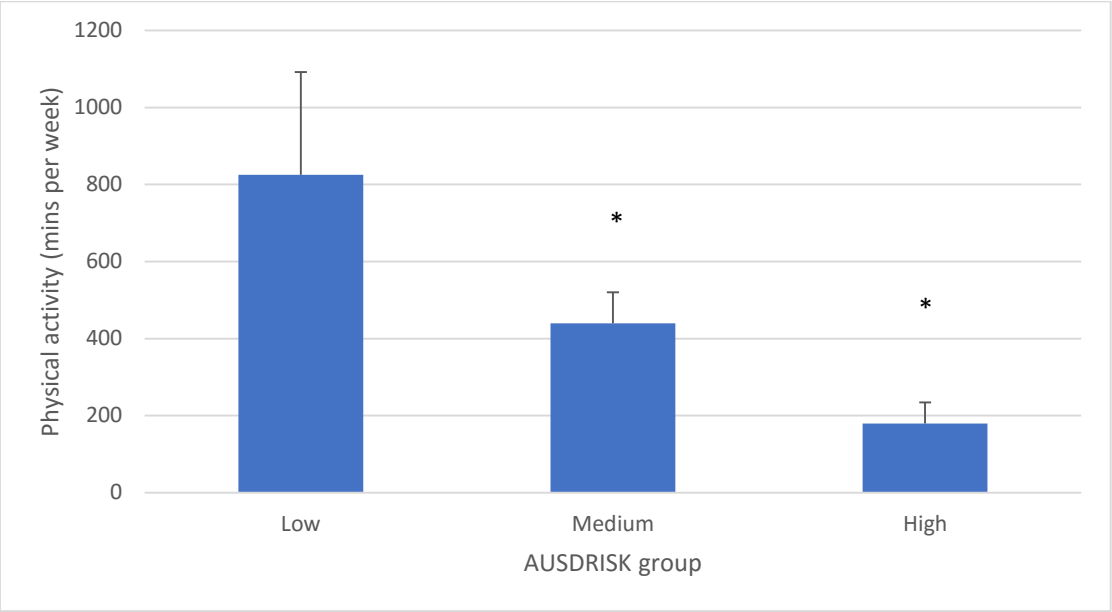


Figure 1. Mean weekly physical activity in Diabetes risk groups[a] (n = 228, mean ± SEM)

[a)]Categorised using the AUSDRISK tool (Chen et al., 2010)

*denotes significant difference from low-risk group at the $p<0.05$ level.

Spearman's correlations were performed as the data was non-parametric. There was a medium sized negative correlation between AUSDRISK score and physical activity ($r(226) = -0.32$, $p < 0.01$) (Appendix 5.2). Using a linear regression analysis a significant association between AUSDRISK score and physical activity was found ($F(1,226) = 6.46$, $p = 0.012$) (Appendix 5.3). Participants' physical activity decreased by 57 minutes (per week) for every point increase in the AUSDRISK questionnaire.

*Sugar sweetened beverages*

A Kruskall-Wallis ANOVA was conducted to compare SSB (kcal per day) between the T2DM risk groups (Appendix 6.1). There was not a significant effect of SSB at the $p < 0.05$ level for the three risk groups [$F(2, 187) = 3.28$, $p = 0.194$]. There was also no significant correlation or linear regression association between AUSDRISK score and SSB intake ($p > 0.05$) (Appendix 6.2-6.3). The low-risk group consumed 182 ± 230 kcal, medium-risk group consumed 203 ± 217 kcal, and the high-risk group consumed 208 ± 170 kcal of SSB daily.

*Total fat*

A Kruskall-Wallis ANOVA was conducted to compare total fat (DINE score) between the T2DM risk groups (Appendix 7.1). There was not a significant effect of total fat intake at the $p < 0.05$ level for the three risk groups [$F(2, 216) = 2.89$, $p = 0.236$]. There was also no significant correlation or linear regression association between AUSDRISK score and total fat intake ($p > 0.05$) (Appendix 7.2-7.3).

By groups, 52.2% of participants fell into low-fat consumption, 28.1% into medium-fat consumption and 15.4% into high-fat consumption.

*Unsaturated fat*

A Kruskall-Wallis ANOVA was conducted to compare unsaturated fat intake (DINE score) between the T2DM risk groups (Appendix 8.1). There was not a significant effect of unsaturated fat intake at the $p < 0.05$ level for the three risk groups [$F(2, 206) = 5.47$, $p = 0.065$] (Figure 2).

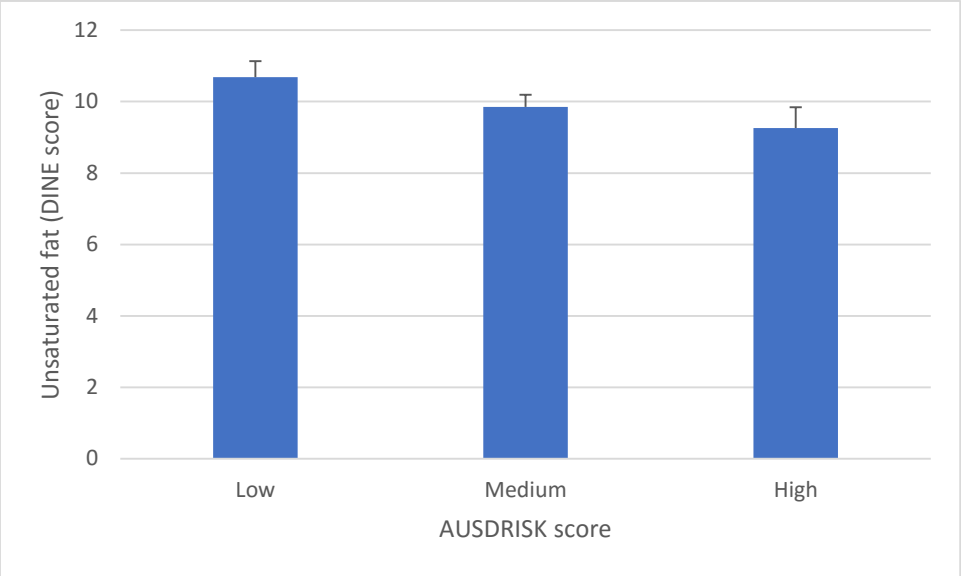


Figure 2. Unsaturated fat intake[a] between Diabetes risk groups[b] (n= 208, mean ± SEM)

[a)]calculated using the DINE tool (Roe et al., 1994)

[b)]Categorised using the AUSDRISK tool (Chen et al., 2010)

However, a significant small negative relationship was observed between AUSDRISK score and unsaturated fat (r(206)= -0.176, $p$ = 0.011) (Appendix 8.2). Linear regression found no association between unsaturated fat intake and AUSDRISK score (Appendix 8.3)

By groups, 12.7% were low unsaturated fat consumers, 24.6% medium unsaturated fat consumers and 53.9% high unsaturated fat consumers.

Discussion

Sixteen percent of individuals were deemed at high risk of developing T2DM in the next five years. On average, these participants were older and had a greater BMI than the low or medium-risk group ($p$ < 0.01). This highlights an important subgroup group of individuals who are vulnerable to accelerating the progress of the disease. This is

especially important as initial diagnosis of T2DM is most commonly in the 45-64 age group (Centres for Disease Control and Prevention, 2017). Therefore, this provides opportunities for interventions to lower that risk and prevent further progression of T2DM.

*Physical activity*

Physical activity was found to be an important predictor of AUSDRISK score, with more PA predicting a lower score. This is supported in the literature by numerous systematic reviews (Smith, Crippa, Woodcock, & Brage, 2016; Aune, Norat, Leitzmann, Tonstad, & Vatten, 2015; Franz, Boucher, Rutten-Ramos, & VanWormer, 2015).

The mechanisms behind the attenuated risk have previously been eluded to indirectly through its role in weight loss (Swift, Johannsen, Lavie, Earnest & Church, 2014), and direct favourable impacts on glucose and lipid metabolism (Sylow, Kleinert, Richter & Jensen, 2017, Colberg et al., 2010). Exercise can both acutely activate glucose transport and improve insulin sensitivity for up to 48 h after exercise (Sylow et al., 2017). Chronic beneficial effects of exercise positively affecting T2DM risk include increased muscle mass, improved insulin action, glucose control, fat oxidation and storage (Colberg et al., 2010).

Caution should be taken when interpreting the results. The average physical activity in the low risk group was 825 minutes, over three times the recommended activity. Also, the range of physical activity was large. Large outliers are likely due to incorrectly completing questionnaires, either intentionally or unintentionally. Excluding anomalies could have eradicated this problem, however may have reduced the power of the

analysis. However, due to clear outliers, it appears reasonable to overlook the absolute values and focus more on the trend in results.

Additionally, some individuals regarded their working day as physical activity, which is impossible to verify. Conversely, other individuals may have not regarded their occupation as a means of physical activity, causing ambiguity in results. More objective methods such as accelerometers would provide additional, more accurate information regarding an individuals' physical activity level throughout the course of a day. Therefore, it would not be unreasonable to overlook the absolute values. Despite these limitations, the data clearly demonstrates a trend of more physical activity being of greater benefit.

Additionally, it was assumed that a blank response in the questionnaire meant the individual participated in no physical activity. However, this could be due to individuals merely not answering the question. Further instructions should perhaps be given to clarify how to fill in the questionnaire correctly.

*Sugar Sweetened Beverages*

There was no statistically significant difference in SSB between risk groups, and no linear relationship was observed with AUSDRISK score. This contrasts with other research which has found SSB positively associated with T2DM (Wu et al., 2014; Imamura et al., 2015; Mozaffarian, 2016). This could be due to the younger population in which this research was conducted skewing the results (35.3 years ± 11.7 years); whereas most studies recruit individuals over the age of 40 (Imamura et al., 2015). This is supported by SSB consumption being highest in young adults (Singh et al., 2015).

Due to the heavier weighting of age in relation to AUSDRISK score, this could be confounding the results, as SSB is similar between all three groups.

The average Australian consume 60 g of free sugar (defined as added sugar plus the sugar component of honey and fruit juices) per day equating to approximately 232 kcal (Australian Bureau of Statistics, 2016b). The results from the present study are lower than this, suggesting individuals may have underreported their consumption. However, such large ranges in consumption make it difficult for conclusions to be drawn.

Other potential explanations for a non-significant finding is the potentially contradictory beneficial effect of coffee and tea (Ding, Bhupathiraju, Chen, Van Dam & Hu, 2014; Bhupathiraju et al., 2012). A systematic review by Ding et al. (2014) concluded a dose-response relationship between coffee consumption and T2DM risk with one cup per day equating to a RR of 0.92 (95% CI 0.90, 0.94), and six cups per day conferring a greater benefit (RR 0.67 95% CI 0.61, 0.74). This dose-response relationship as observed with caffeinated and decaffeinated coffee. The mechanisms behind this are largely unknown, and appears to be a complex interaction, of which warrants further investigation.

Also, SSB only reflects part of the diet. The average Australian intake of total sugar is 102 g per day, or 406 kcal (Australian Bureau of Statistics, 2016b). This study provides no information regarding additional sources of sugar in the diet.

*Total fat*

Whilst no significant relationships were found with total fat, this is perhaps not surprising as research suggests the composition of fat in the diet is more important

than total fat consumption (Ley, Hamdy, Mohan & Hu, 2014; Ortega et al., 2017). This is because saturated and unsaturated fat have contradictory effects, which may negate any effect of total fat.

When coding total fat into low, medium and high fat groups, over half of participants fell into the low-fat group, which corresponds to < 35% energy intake or < 83 g/day (Roe et al., 1994). This is consistent with the average Australian consumption of fat constituting 31% of energy intake (Australian Bureau of Statistics, 2014). However, underreporting cannot be excluded.

*Unsaturated fat*

A linear correlation was the only significant finding with AUSDRISK score, albeit only a small effect (Cohen, 1992). The non-significant linear regression results suggest this is not an independent risk factor.

Interestingly, no significant difference was found between risk groups ($p = 0.065$). This seems to contradict evidence from a recent pooled analysis of 20 studies concluding increased consumption of unsaturated fat results in a lower T2DM risk (Wu et al., 2017).

However, this non-significant finding could be explained through several potential reasons. Due to the limited questions pertaining to unsaturated fat intake; questions incorporating more food groups such as fish and nuts may have resulted in different results. Secondly, the proportion of energy from unsaturated fat has not been addressed, and absolute numbers may not accurately reflect dietary composition of fat

intake. Thirdly, the relatively small sample size may not be sufficient to identify subtle differences.

Despite the non-significant finding, there is a trend for the average consumption of unsaturated fat to decrease with an increase in risk group. This therefore still has important implications when intervening and making recommendations to this population to reduce their T2DM risk.

*AUSDRISK questionnaire*

A potential reason for a majority of non-significant findings could be due to limitations associated with the method employed to define risk of T2DM. This questionnaire is heavily relied upon genetic factors (age, gender, ethnicity, family history), as a high-risk score can be achieved solely by being male, > 65 years old and being an Aboriginal and Torres Strait Islander People. Only 3 points on the questionnaire are regarding diet and PA, representing less than 8% of the total possible score. This therefore may underestimate the lifestyle effects associated with T2DM. However, only having a few points for lifestyle factors may prevent misrepresentative relationships being observed between lifestyle variables and AUSDRISK score purely due to the weighting of the questions in calculating AUSDRISK score.

Additionally, this population in this study varies significantly from the population used to validate the questionnaire which had 55.1% females and an average age of 51.5 years, and included 0.8% from Indigenous populations (Dunstan et al., 2002). Conversely, this population was 99% male, average age of 35.6 years and included 1.2% from Indigenous populations. Both studies underrepresent the Indigenous

population which have a prevalence of 2% in Australia (Australian Bureau of Statistics, 2016a). Also, this study is based on a cohort from 1999-2000, and may not reflect demographic characteristics of individuals in Australia today. Therefore, this questionnaire may not be valid in assessing T2DM risk in this population. Further research should explore developing a questionnaire with more current Australian demographic characteristics.

Individuals defined as high-risk of developing T2DM had a significantly higher BMI than the low or medium-risk groups. Being overweight or obese is the single largest predictor of T2DM (Kharroubi, & Darwish, 2015), however BMI is not the most robust method of assessing this (Kolb & Martin, 2017). Waist circumference is a preferable as it is a better indicator of visceral fat and is not confounded by muscle mass (Kolb & Martin, 2017). However, comparisons with waist circumference were not done. This is due to the potential close relationship between the variables, as waist circumference is a question in the AUSDRISK questionnaire, with a possible score of 7, equating to almost 20% of the total score. This could suggest a linear relationship would exist between waist circumference and AUSDRISK purely due to the weighting of waist circumference in determining AUSDRISK score. It therefore would be difficult to accurately examine the relationship of waist circumference using this questionnaire as a method of determining T2DM risk.

*General limitations*

This is a cross sectional study, and therefore only provides a snapshot of an individuals' habitual behaviour. A longitudinal study would provide more in-depth information

regarding potential variations in habitual behaviours e.g. seasonal changes in diet and PA.

Secondly, questionnaires are self-reported and inherently limited by their subjective nature, and liability to underreporting and response bias (Probst & Zammit, 2016). Additionally, individuals tend to misreport certain variables: height and PA are often over-reported and weight and dietary intake is commonly underreported (Ainsworth, Cahalin, Buman, & Ross, 2015). This reduces the validity of the responses and can ultimately lead to incorrect conclusions to be drawn.

Thirdly, 22 incomplete questionnaires were excluded for analysis of AUSDRISK score with further exclusions for comparisons with SSB (n = 39), total fat (n = 10), unsaturated fat (n = 20). Incomplete was regarded as two or more questions not answered. Therefore, the scores obtained could still be underestimations as participants were included if one question was unanswered. Consciously not answering a single question could be intentional and may reflect social desirability bias (Probst & Zammit, 2016) i.e. purposefully not answering a question deemed to be socially undesirable.

*Future directions*

Future research should investigate long term lifestyle behaviours within larger samples of this population, using more objective measures such as accelerometers and weighed food diaries. However, it is recognised these are very time intensive measures relying on great adherence, which may not be realistically achievable in a population who work long hours (Lingard & Turner, 2018).

Further research could also look at the incidence of T2DM in this population by following this group over time, and objectively assessing incidence of T2DM as the main outcome.

Furthermore, identification of a group of high-risk individuals requires exploration of interventions to delay or prevent T2DM within these individuals and assessing the efficacy of such interventions.

*Conclusion*

This study identified 16% of individuals within our population at high-risk of developing T2DM. There was a tendency for these individuals to be older, have a greater BMI, participate in less PA and consume less unsaturated fat. This provides an insight into areas to target for interventions in these individuals.

All risk groups had an average BMI that reflects an overweight population, the single largest predictor of T2DM (Kharroubi, & Darwish, 2015). Therefore, even the low-risk group should be encouraged to maintain a healthy weight. The results of this study should be used to aid the development of strategies to delay or reverse the progression of T2DM for all individuals, but particularly in these high-risk individuals. Reducing the risk of T2DM would have positive impacts on the individual: enhanced quality of life, more metabolically healthy (Samson et al., 2016), and for the economy: less sick days, later retirement, higher productivity (Schofield et al., 2017).

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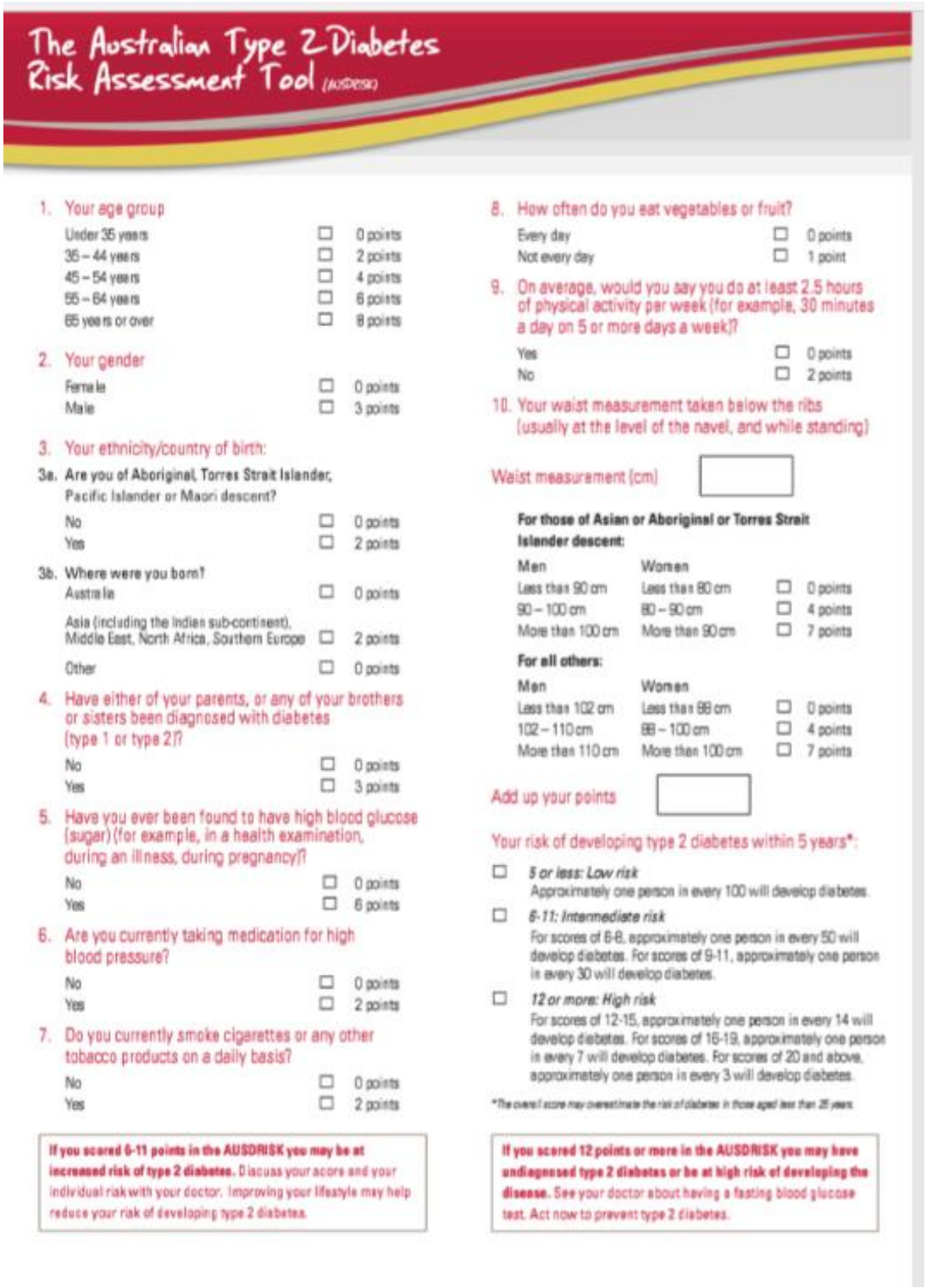
# Appendix

## Appendix 1

*Questionnaires*

AUSDRISK tool (Chen et al., 2010).



The Australian Type 2 Diabetes Risk Assessment Tool (AUSDRISK)

1. Your age group

| | |
|---|---|
| Under 35 years | ☐ 0 points |
| 35 – 44 years | ☐ 2 points |
| 45 – 54 years | ☐ 4 points |
| 55 – 64 years | ☐ 6 points |
| 65 years or over | ☐ 8 points |

2. Your gender

| | |
|---|---|
| Female | ☐ 0 points |
| Male | ☐ 3 points |

3. Your ethnicity/country of birth:

3a. Are you of Aboriginal, Torres Strait Islander, Pacific Islander or Maori descent?

| | |
|---|---|
| No | ☐ 0 points |
| Yes | ☐ 2 points |

3b. Where were you born?

| | |
|---|---|
| Australia | ☐ 0 points |
| Asia (including the Indian sub-continent), Middle East, North Africa, Southern Europe | ☐ 2 points |
| Other | ☐ 0 points |

4. Have either of your parents, or any of your brothers or sisters been diagnosed with diabetes (type 1 or type 2)?

| | |
|---|---|
| No | ☐ 0 points |
| Yes | ☐ 3 points |

5. Have you ever been found to have high blood glucose (sugar) (for example, in a health examination, during an illness, during pregnancy)?

| | |
|---|---|
| No | ☐ 0 points |
| Yes | ☐ 6 points |

6. Are you currently taking medication for high blood pressure?

| | |
|---|---|
| No | ☐ 0 points |
| Yes | ☐ 2 points |

7. Do you currently smoke cigarettes or any other tobacco products on a daily basis?

| | |
|---|---|
| No | ☐ 0 points |
| Yes | ☐ 2 points |

8. How often do you eat vegetables or fruit?

| | |
|---|---|
| Every day | ☐ 0 points |
| Not every day | ☐ 1 point |

9. On average, would you say you do at least 2.5 hours of physical activity per week (for example, 30 minutes a day on 5 or more days a week)?

| | |
|---|---|
| Yes | ☐ 0 points |
| No | ☐ 2 points |

10. Your waist measurement taken below the ribs (usually at the level of the navel, and while standing)

Waist measurement (cm) ☐

**For those of Asian or Aboriginal or Torres Strait Islander descent:**

| Men | Women | |
|---|---|---|
| Less than 90 cm | Less than 80 cm | ☐ 0 points |
| 90 – 100 cm | 80 – 90 cm | ☐ 4 points |
| More than 100 cm | More than 90 cm | ☐ 7 points |

**For all others:**

| Men | Women | |
|---|---|---|
| Less than 102 cm | Less than 88 cm | ☐ 0 points |
| 102 – 110 cm | 88 – 100 cm | ☐ 4 points |
| More than 110 cm | More than 100 cm | ☐ 7 points |

Add up your points ☐

Your risk of developing type 2 diabetes within 5 years*:

☐ *5 or less: Low risk*
Approximately one person in every 100 will develop diabetes.

☐ *6-11: Intermediate risk*
For scores of 6-8, approximately one person in every 50 will develop diabetes. For scores of 9-11, approximately one person in every 30 will develop diabetes.

☐ *12 or more: High risk*
For scores of 12-15, approximately one person in every 14 will develop diabetes. For scores of 16-19, approximately one person in every 7 will develop diabetes. For scores of 20 and above, approximately one person in every 3 will develop diabetes.

*The overall score may overestimate the risk of diabetes in those aged less than 25 years.

**If you scored 6-11 points in the AUSDRISK you may be at increased risk of type 2 diabetes.** Discuss your score and your individual risk with your doctor. Improving your lifestyle may help reduce your risk of developing type 2 diabetes.

**If you scored 12 points or more in the AUSDRISK you may have undiagnosed type 2 diabetes or be at high risk of developing the disease.** See your doctor about having a fasting blood glucose test. Act now to prevent type 2 diabetes.

BEVQ-15 questionnaire (Hedrick et al., 2012, adapted by research dietician Katherine Markwell)

**The purpose of this section is to get an idea of your usual eating and drinking habits.** For the past month, please indicate your response for each beverage type by choosing both "how often" and "how much each time".

*1. Indicate how often you drank the following beverages, for example, if you drank 5 cups of tea per week, mark 4-6 times per week.*

*2. Indicate the approximate amount of beverage you drank each time, for example, if you drank one cup of water each time, mark one cup under "how much each time".*

If 0, go to next drink

| | How often (mark one) | | | | | | | How much each time (mark one) | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Never or less than 1/week | Once a week | 2-3 times a week | 4-6 times a week | Once a day | Twice a day | Three or more times a day | Standard glass (120 ml) | Small bottle/cup (250 ml) | Regular can (375 ml) | Large bottle/can (675 ml) | Bottle or carton (1 litre) |
| **Soft drinks, regular (that is, not diet, not caffeinated e.g. lemonade)** | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 0 | 1 | 2 | 3 | 4 |
| **Caffeinated Soft Drinks (e.g. Coke)** | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 0 | 1 | 2 | 3 | 4 |
| **Energy Drinks (e.g. Mother, Red Bull, V)** | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 0 | 1 | 2 | 3 | 4 |
| **Diet soft drinks (e.g. Sprite Zero)** | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 0 | 1 | 2 | 3 | 4 |
| **Sports Drinks (e.g. Gatorade, Powerade)** | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 0 | 1 | 2 | 3 | 4 |
| **Beers, Ales, Wine, Cider** | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 0 | 1 | 2 | 3 | 4 |
| **Non-Alcoholic or Light Beer** | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 0 | 1 | 2 | 3 | 4 |
| **Spirits (shots, rum, whiskey, etc.)** | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 0 | 1 | 2 | 3 | 4 |
| **Tea or Coffee** | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 0 | 1 | 2 | 3 | 4 |
| **Full cream milk (plain)** | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 0 | 1 | 2 | 3 | 4 |
| **Reduced fat or skimmed milk** | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 0 | 1 | 2 | 3 | 4 |
| **Flavoured milk (eg. Breaka, Iced Coffee)** | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 0 | 1 | 2 | 3 | 4 |

**If your drink tea or coffee, how many rounded teaspoons of sugar per drink?**

0 1 2 3 4 5 6

DINE tool (Roe et al., 1994)

About how many **servings per week** do you eat of the following foods?
(Please tick one box on each line)

| | | None | Less than 1 a week | 1 to 2 a week | 3 to 5 a week | 6 or more a week |
|---|---|---|---|---|---|---|
| 13. | Cheese (any except cottage) | | | | | |
| 14. | Beefburgers or sausages | | | | | |
| 15. | Beef, pork, or lamb (for vegetarians: nuts) | | | | | |
| 16. | Bacon, meat pie, processed meat | | | | | |
| 17. | Chicken or turkey | | | | | |
| 18. | Fish (NOT fried fish) | | | | | |
| 19. | ANY fried food: fried fish, chips, cooked breakfast, samosas | | | | | |
| 20. | Cakes, pies, puddings, pastries | | | | | |
| 21. | Biscuits, chocolate, or crisps | | | | | |
| | | None | Less than 1 a week | 1 to 2 a week | 3 to 5 a week | 6 or more a week |

About how much of the following types of milk do you yourself use **in a day**, for example in cereal, tea, or coffee? (Please tick one box on each line)

| | **Milks** | None | Less than half a cooking cup | About half a cooking cup | About one cooking cup | Two cooking cups or more |
|---|---|---|---|---|---|---|
| 22. | Full cream | | | | | |
| 23. | Semi-skimmed, lite, 2 % fat (e.g. smarter white milk, trim) | | | | | |
| 24. | Skimmed,(no fat, fat free, Shape, Heart Active) | | | | | |

About how many **rounded teaspoons per day** do you usually use of the following types of spreads, for example on bread, sandwiches, toast, potatoes, or vegetables?

| | **Spreads** | None | 1 a day | 2 a day | 3 a day | 4 a day | 5 a day | 6 a day | 7 or more |
|---|---|---|---|---|---|---|---|---|---|
| 25. | **Regular margarine or butter** or **Reduced fat spread** such as sunflower, canola or olive spread, Flora, ,Nutulex, Tablelands, | | | | | | | | |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Meadow Lea, Devondale Spreadable | | | | | | | | |
| 26. | **Low fat spread** such as Flora Light, Nutulex light, Devondale light Flora Pro-activ, | | | | | | | | |

| What type of fat do you usually use for the following purposes? (Please tick one box on each line) | | | | | | |
|---|---|---|---|---|---|---|
| | | **Butter, lard, or dripping** | **Solid cooking fat** (copha) **Half-fat butter** (Devondale Spreadable) **Hard margarine** (cooking margarine) | **Soft margarine** (sunflower, canola) **Reduced fat spread** (Devondale Light) | **Vegetable oil** (olive, sunflower, canola oil) or **Low fat spread** (Flora Light, Flora Pro-activ) | **No fat used** |
| 27. | On bread and vegetables | | | | | |
| 28. | For frying | | | | | |
| 29. | For baking or cooking | | | | | |

Adapted short IPAQ (Craig et al., 2003)

**Think about an average week in the past months. Please indicate how many days per week you performed the following activities, how much time on average you were engaged in this, and (if applicable) how strenuous this activity was for you.**

**Sports and leisure activites** ***Please complete all columns***

**(Please write in which activities you participate in, e.g. gym, martial arts, running, gardening)**

**SKIP IF NOT APPLICABLE**

| | Times per week | | Intensity... |
|---|---|---|---|
| 1. ..... | ① ② ③ ④ ⑤ ⑥ ⑦ | ☐ minutes per week | ① slow ② moderate ③ fast |
| 2. ..... | ① ② ③ ④ ⑤ ⑥ ⑦ | ☐ minutes per week | ① slow ② moderate ③ fast |
| 3. ..... | ① ② ③ ④ ⑤ ⑥ ⑦ | ☐ minutes per week | ① slow ② moderate ③ fast |
| 4. ..... | ① ② ③ ④ ⑤ ⑥ ⑦ | ☐ minutes per day | ① slow ② moderate ③ fast |

Additional demographic questions

**Approximate weight.** ***You can mark either kgs or stones/pounds.***

☐ kgs ***OR*** ☐ stones ☐ pounds

**Approximate height.**

☐ cms ***OR*** ☐ feet ☐ inches

**Approximate waist circumference taken at your belly button**
***(not your pant size)***

☐ cms ***OR*** ☐ inches

Sex: Male or Female

Age: _____ years

## *SPSS tables*

## Appendix 2

Descriptive statistics for participants included in AUSDRISK analysis

**Descriptives**

| | | | Statistic | Std. Error |
|---|---|---|---|---|
| Age | Mean | | 35.3295 | .88993 |
| | 95% Confidence Interval for Mean | Lower Bound | 33.5729 | |
| | | Upper Bound | 37.0861 | |
| | 5% Trimmed Mean | | 35.2595 | |
| | Median | | 34.0000 | |
| | Variance | | 137.013 | |
| | Std. Deviation | | 11.70525 | |
| | Minimum | | -1.00 | |
| | Maximum | | 61.00 | |
| | Range | | 62.00 | |
| | Interquartile Range | | 17.00 | |
| | Skewness | | .024 | .185 |
| | Kurtosis | | -.101 | .367 |
| BMI | Mean | | 27.1463 | .35389 |
| | 95% Confidence Interval for Mean | Lower Bound | 26.4478 | |
| | | Upper Bound | 27.8448 | |
| | 5% Trimmed Mean | | 26.7733 | |
| | Median | | 26.3158 | |
| | Variance | | 21.666 | |
| | Std. Deviation | | 4.65466 | |
| | Minimum | | 17.96 | |
| | Maximum | | 56.17 | |
| | Range | | 38.21 | |
| | Interquartile Range | | 4.12 | |
| | Skewness | | 3.008 | .185 |
| | Kurtosis | | 15.565 | .367 |

## Appendix 3

Normal distribution for AUSDRISK score

**Tests of Normality**

| | Kolmogorov-Smirnov [a] | | | Shapiro-Wilk | | |
|---|---|---|---|---|---|---|
| | Statistic | df | Sig. | Statistic | df | Sig. |
| AUSDRISK_score | .128 | 228 | .000 | .930 | 228 | .000 |

a. Lilliefors Significance Correction

## Appendix 4

### *Appendix 4.1*

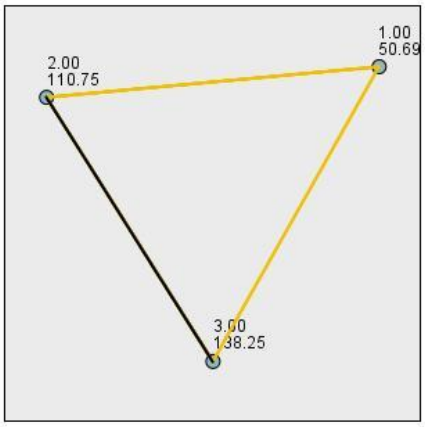
Kruskall-Wallis for age between AUSDRISK groups with post-hoc analysis (Mann-Whitney tests)

**Hypothesis Test Summary**

| | Null Hypothesis | Test | Sig. | Decision |
|---|---|---|---|---|
| 1 | The distribution of Age is the same across categories of AUSDRISK_Group. | Independent-Samples Kruskal-Wallis Test | .000 | Reject the null hypothesis. |

Asymptotic significances are displayed. The significance level is .05.

**Pairwise Comparisons of AUSDRISK_Group**




Each node shows the sample average rank of AUSDRISK_Group.

| Sample1-Sample2 | Test Statistic | Std. Error | Std. Test Statistic | Sig. | Adj.Sig. |
|---|---|---|---|---|---|
| 1.00-2.00 | -60.052 | 9.141 | -6.570 | .000 | .000 |
| 1.00-3.00 | -87.557 | 12.885 | -6.795 | .000 | .000 |
| 2.00-3.00 | -27.505 | 11.841 | -2.323 | .020 | .061 |

Each row tests the null hypothesis that the Sample 1 and Sample 2 distributions are the same.
Asymptotic significances (2-sided tests) are displayed. The significance level is .05.
Significance values have been adjusted by the Bonferroni correction for multiple tests.

*Appendix 4.2*

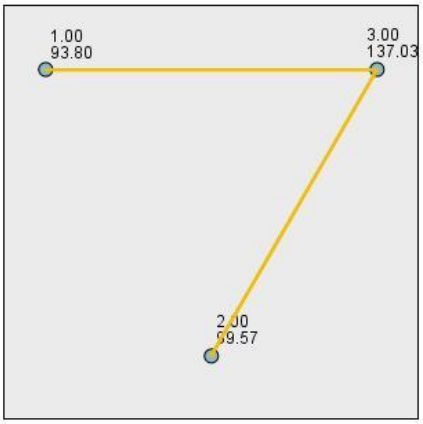
Kruskall-Wallis for BMI between AUSDRISK groups with post-hoc analysis (Mann-Whitney tests)

**Hypothesis Test Summary**

| | Null Hypothesis | Test | Sig. | Decision |
|---|---|---|---|---|
| 1 | The distribution of BMI is the same across categories of AUSDRISK_Group. | Independent-Samples Kruskal-Wallis Test | .003 | Reject the null hypothesis. |

Asymptotic significances are displayed. The significance level is .05.

**Pairwise Comparisons of AUSDRISK_Group**




Each node shows the sample average rank of AUSDRISK_Group.

| Sample1-Sample2 | Test Statistic | Std. Error | Std. Test Statistic | Sig. | Adj.Sig. |
|---|---|---|---|---|---|
| 1.00-2.00 | -5.779 | 9.456 | -.611 | .541 | 1.000 |
| 1.00-3.00 | -43.237 | 13.148 | -3.288 | .001 | .003 |
| 2.00-3.00 | -37.458 | 12.075 | -3.102 | .002 | .006 |

Each row tests the null hypothesis that the Sample 1 and Sample 2 distributions are the same.
Asymptotic significances (2-sided tests) are displayed. The significance level is .05.
Significance values have been adjusted by the Bonferroni correction for multiple tests.

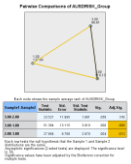
## Appendix 5 - Physical activity

*Appendix 5.1* Kruskall-Wallis ANOVA for physical activity between AUSDRISK groups and post-hoc analysis (Mann-Whitney tests)

**Hypothesis Test Summary**

| | Null Hypothesis | Test | Sig. | Decision |
|---|---|---|---|---|
| 1 | The distribution of PA is the same across categories of AUSDRISK_Group. | Independent-Samples Kruskal-Wallis Test | .000 | Reject the null hypothesis. |

Asymptotic significances are displayed. The significance level is .05.



## *Appendix 5.2* Spearman correlation for AUSDRISK and physical activity

**Correlations**

| | | | AUSDRISK_score | PA |
|---|---|---|---|---|
| Spearman's rho | AUSDRISK_score | Correlation Coefficient | 1.000 | -.320 ** |
| | | Sig. (2-tailed) | . | .000 |
| | | N | 228 | 228 |
| | PA | Correlation Coefficient | -.320 ** | 1.000 |
| | | Sig. (2-tailed) | .000 | . |
| | | N | 228 | 228 |

**. Correlation is significant at the 0.01 level (2-tailed).

## *Appendix 5.3* Linear regression for AUSDRISK and physical activity

**Model Summary**

| Model | R | R Square | Adjusted R Square | Std. Error of the Estimate |
|---|---|---|---|---|
| 1 | .167 a | .028 | .024 | 1357.23308 |

a. Predictors: (Constant), AUSDRISK_score

**ANOVA a**

| Model | | Sum of Squares | df | Mean Square | F | Sig. |
|---|---|---|---|---|---|---|
| 1 | Regression | 11906398.68 | 1 | 11906398.68 | 6.464 | .012 b |
| | Residual | 416310448.4 | 226 | 1842081.630 | | |
| | Total | 428216847.1 | 227 | | | |

a. Dependent Variable: PA

b. Predictors: (Constant), AUSDRISK_score

**Coefficients a**

| Model | | Unstandardized Coefficients | | Standardized Coefficients | | |
|---|---|---|---|---|---|---|
| | | B | Std. Error | Beta | t | Sig. |
| 1 | (Constant) | 968.550 | 201.325 | | 4.811 | .000 |
| | AUSDRISK_score | -57.407 | 22.580 | -.167 | -2.542 | .012 |

a. Dependent Variable: PA

## Appendix 6 – Sugar sweetened beverages

*Appendix 6.1* Kruskall-Wallis ANOVA for SSB

**Hypothesis Test Summary**

| | Null Hypothesis | Test | Sig. | Decision |
|---|---|---|---|---|
| 1 | The distribution of SSB_kcal is the same across categories of AUSDRISK_Group. | Independent-Samples Kruskal-Wallis Test | .194 | Retain the null hypothesis. |

Asymptotic significances are displayed. The significance level is .05.

*Appendix 6.2* Spearman correlation for AUSDRISK and SSB

**Correlations**

| | | | AUSDRISK_score | SSB_kcal |
|---|---|---|---|---|
| Spearman's rho | AUSDRISK_score | Correlation Coefficient | 1.000 | .100 |
| | | Sig. (2-tailed) | . | .170 |
| | | N | 228 | 189 |
| | SSB_kcal | Correlation Coefficient | .100 | 1.000 |
| | | Sig. (2-tailed) | .170 | . |
| | | N | 189 | 189 |

*Appendix 6.3* Linear regression between AUSDRISK and SSB

**Coefficients[a]**

| Model | | Unstandardized Coefficients | | Standardized Coefficients | | |
|---|---|---|---|---|---|---|
| | | B | Std. Error | Beta | t | Sig. |
| 1 | (Constant) | 177.090 | 35.045 | | 5.053 | .000 |
| | AUSDRISK_score | 2.551 | 3.885 | .048 | .657 | .512 |

a. Dependent Variable: SSB_kcal

## Appendix 7 – Total fat

*Appendix 7.1* Kruskall-Wallis ANOVA for total fat

**Hypothesis Test Summary**

| | Null Hypothesis | Test | Sig. | Decision |
|---|---|---|---|---|
| 1 | The distribution of DINE_total is the same across categories of AUSDRISK_Group. | Independent-Samples Kruskal-Wallis Test | .236 | Retain the null hypothesis. |

Asymptotic significances are displayed. The significance level is .05.

*Appendix 7.2* Spearman correlation for AUSDRISK and total fat

**Correlations**

| | | | AUSDRISK_score | DINE_total |
|---|---|---|---|---|
| Spearman's rho | AUSDRISK_score | Correlation Coefficient | 1.000 | .066 |
| | | Sig. (2-tailed) | . | .332 |
| | | N | 228 | 218 |
| | DINE_total | Correlation Coefficient | .066 | 1.000 |
| | | Sig. (2-tailed) | .332 | . |
| | | N | 218 | 218 |

*Appendix 7.3* Linear regression for AUSDRISK and total fat

**Coefficients[a]**

| Model | | Unstandardized Coefficients B | Unstandardized Coefficients Std. Error | Standardized Coefficients Beta | t | Sig. |
|---|---|---|---|---|---|---|
| 1 | (Constant) | 29.596 | 1.991 | | 14.865 | .000 |
| | AUSDRISK_score | .110 | .222 | .034 | .493 | .623 |

a. Dependent Variable: DINE_total

## Appendix 8 – Unsaturated fat

*Appendix 8.1* Kruskall-Wallis ANOVA for unsaturated fat

**Hypothesis Test Summary**

| | Null Hypothesis | Test | Sig. | Decision |
|---|---|---|---|---|
| 1 | The distribution of DINE_unsat is the same across categories of AUSDRISK_Group. | Independent-Samples Kruskal-Wallis Test | .065 | Retain the null hypothesis. |

Asymptotic significances are displayed. The significance level is .05.

*Appendix 8.2* Spearman correlation for AUSDRISK and unsaturated fat

**Correlations**

| | | | AUSDRISK_score | DINE_unsat |
|---|---|---|---|---|
| Spearman's rho | AUSDRISK_score | Correlation Coefficient | 1.000 | -.176 * |
| | | Sig. (2-tailed) | . | .011 |
| | | N | 228 | 208 |
| | DINE_unsat | Correlation Coefficient | -.176 * | 1.000 |
| | | Sig. (2-tailed) | .011 | . |
| | | N | 208 | 208 |

*. Correlation is significant at the 0.05 level (2-tailed).

*Appendix 8.3* Linear regression for AUSDRISK and unsaturated fat

**Coefficients[a]**

| Model | | Unstandardized Coefficients | | Standardized Coefficients | | |
|---|---|---|---|---|---|---|
| | | B | Std. Error | Beta | t | Sig. |
| 1 | (Constant) | 10.926 | .544 | | 20.072 | .000 |
| | AUSDRISK_score | -.116 | .061 | -.132 | -1.917 | .057 |

a. Dependent Variable: DINE_unsat